

Effect of individual environmental heat stress variables on training and recovery in professional team sport

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1 Running Head: Thermoregulation and team-sport training

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3 **Effect of independent environmental heat stress variables on**
4 **training and recovery in professional team sport**

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30 **Abstract**

31 **Purpose:** Exercise in hot environments increases body temperature and
32 thermoregulatory strain. However, little is known regarding the magnitude
33 of effect that ambient temperature (T_a), relative humidity (RH) and solar
34 radiation (SR) individually have on team sport athletes. We aimed to
35 determine the effect of these individual heat stress variables on team-sport
36 training performance and recovery. **Methods:** Professional Australian
37 Rules Football (ARF) players ($n=45$) undertook eight-weeks pre-season
38 training producing a total of 579 outdoor field-based observations with T_a ,
39 RH and SR recorded at every training session. External load (distance
40 covered, $\text{m}\cdot\text{min}^{-1}$; percent high speed running $>14.4 \text{ km}\cdot\text{h}^{-1}$; %HSR) was
41 collected via a global positioning system. Internal load (ratings of
42 perceived exertion (RPE), heart rate (HR)), and recovery (subjective
43 ratings of wellbeing and heart rate variability (rMSSD)) were monitored
44 throughout the training period. Mixed effects linear models analysed
45 relationships between variables using standardised regression coefficients.
46 **Results:** Increasing SR exposure was associated with reduced distance
47 covered ($-19.7 \text{ m}\cdot\text{min}^{-1}$, $\beta=-0.909$, $p<0.001$), %HSR (-10% , $\beta=-0.953$,
48 $p<0.001$) during training, and rMSSD 48 h post-training (-16.9ms , $\beta=-$
49 0.277 , $p=0.019$). Greater RH was associated with decreased %HSR ($-$
50 3.4% , $\beta=-0.319$, $p=0.010$), but increased % duration $>85\%$ HRmax (3.9% ,
51 $\beta=0.260$, $p<0.001$), RPE (1.8AU , $\beta=0.968$, $p<0.001$) and self-reported
52 stress 24 h post-training (-0.11AU , $\beta=-0.24$, $P=0.002$). In contrast, higher
53 T_a was associated with in increased distance covered ($19.7 \text{ m}\cdot\text{min}^{-1}$,
54 $\beta=0.911$, $p<0.001$) and %HSR (3.5% , $\beta=0.338$, $p=0.005$). **Conclusions:**
55 We show the importance of considering the individual factors contributing
56 to thermal load in isolation for team sport athletes, and that SR and RH
57 reduce work capacity during team sport training and have potential to slow
58 recovery between sessions.

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63 **Introduction**

64 Training and competition in the heat can provide additional physiological
65 demands with potential to limit the intensity/duration of exercise, and
66 increase the rating of perceived exertion (RPE) compared with exercise in
67 thermoneutral conditions ¹. Indeed, there is a large body of evidence
68 examining the effects of hot environments whilst undertaking steady-state
69 aerobic exercise ^{1,2}. For example, Galloway and Maughan ³ previously
70 showed that under laboratory conditions of increasing ambient
71 temperature (T_a) with fixed RH there are significant increases in heart rate
72 (HR), core temperature, skin temperature (T_{sk}), sweat rate, RPE and
73 impaired exercise capacity. Similarly, when increasing RH with fixed hot
74 T_a there is also increased T_{sk} , sweat rate, and RPE, and decreased exercise
75 performance ⁴. Finally, Otani and colleagues ⁵ showed that the effects of
76 increased solar radiation (SR) exposure together with constant T_a and RH
77 elevates T_{sk} , reduces the core-to-skin temperature gradient, and
78 subsequently leads to decreased endurance capacity. Accordingly, each
79 heat stress variable contributing to total heat strain may have individual
80 effects with the potential to reduce physical performance in hot conditions.

81 The vast majority of previous research on thermoregulation during
82 exercise in hot environments has focused on the endurance athlete, and
83 there is a paucity of available data examining the effect of exercise in the
84 heat on the team-sport athlete ⁶. Some previous studies have examined
85 physiological changes to heat stress in team sport players, including
86 characterizing responses during match play at various wet bulb globe
87 temperatures ⁷. Many team-sports undertake physical preparation outdoors
88 during summer months at geographical locations characterised by high T_a ,
89 RH and SR ⁶. Repeatedly training in hot environments may promote long-
90 term adaptations such as improved sweat response and decreased
91 cardiovascular strain with the potential to convey a benefit for team sport
92 performance ⁸. Conversely, the acute thermoregulatory responses to
93 exercise in hot conditions may attenuate physical work capacity and the
94 quality of training sessions, while frequent heat exposure during high-

intensity intermittent team sport training may lead to overreaching and adverse effects on athlete wellness and performance⁹.

Understanding the physiological effects of recurring exposure to heat stress during training or competition is necessary to optimise physical preparation and well-being in team sport athletes. Moreover, there is currently no data on varying environmental conditions that consider the individual effects of T_a , RH and SR on external load, internal load and recovery from intermittent, high intensity team-sport training activities. Thus, the primary aim of this study was to determine the individual effects of T_a , RH and SR on professional team-sport training sessions undertaken in hot conditions. Our secondary aim was to establish the individual effect of these heat variables on recovery during the 48 h period following training bouts. We hypothesized that with increasing exposure to each individual heat stress variable, external work completed during training would decrease concomitant with higher internal loads and impaired recovery.

Materials and Methods

Participants

A convenience sample of forty-five professional male athletes (mean \pm standard deviation [SD]; age: 22.9 ± 3.8 yrs, height: 188.4 ± 8.3 cm, body mass: 86.9 ± 9.4 kg, maximal aerobic speed 17.6 ± 0.8 km/h) from one professional football club competing in the Australian Football League participated in this study. Athletes completed a minimum of five weeks pre-season training in a hot environment before the data collection period and were deemed to be heat acclimated. Ethical approval was granted by Bond University Human Research Ethics Committee (FO00007).

Experimental Protocol

Internal and external training loads were captured throughout an eight-week training period of the pre-season preparation phase spanning January and February of the Australian summer. Upon removal of ‘stationary skill’ sessions that were completed during the experimental period, an average

127 16 ± 3 sessions were recorded per player (range: 4 to 20), resulting in 579
128 outdoor field-based training observations in the final analysis. Participants
129 wore the same clothing (singlet and shorts) during each training session.
130 Pre- and post- each training session, body mass (BM) was recorded with
131 participants wearing the same clothing (running shorts) using standardised
132 weighing scales (Excell Precision, New Taipei, Taiwan) to the nearest 100
133 g for calculation of body mass changes from pre- to post-session.

134

135 **External training load**

136 External training loads for each participant were collected and downloaded
137 in accordance with previously described methods ¹⁰. Participants used the
138 same global positioning system (GPS) device (S5, Catapult Sports,
139 Melbourne, Australia) for each session to mitigate inter-unit measurement
140 errors ¹¹. Distance covered per minute (m.min⁻¹) and percent of total
141 distance completed above 14.4 km/h (% high speed running; %HSR) were
142 selected and used in subsequent statistical analyses, in part, to reduce
143 potential issues of multicollinearity. Individual training session data was
144 only included in the analysis if the athlete had completed a minimum of
145 80% of each prescribed training session.

146

147 **Internal training load**

148 Ratings of perceived exertion (RPE) were obtained 10-30 min following
149 the completion of each training session using Borg's CR-10 scale ¹². Heart
150 Rate (HR) data was collected via chest strap HR monitors (T34, Polar
151 Electro, Espoo, Finland). HR data was analysed by quantifying the percent
152 of total duration within specific 'zones' (Zone 3 = 65-74%, Zone 4 = 75-
153 84%, Zone 5 = >85% HR_{max}).

154

155 Individual self-reported ratings of wellbeing were assessed via a
156 psychometric questionnaire on a 10-point Likert scale with 1 representing
157 '*the worst I could possibly feel*' and 10 representing '*the best I could*
158 '*possibly feel*' in accordance with methods described previously ¹³.
159 Objective measures of heart-rate variability (HRV) were assessed upon
160 waking each morning by R-R series recording via photoplethysmography

161 using a valid and reliable, commercially available smartphone application
162 (HRV4Training) ¹⁴. HRV data was subsequently analysed for the root
163 mean sum of the squared differences (rMSSD) between each successive
164 heartbeat on recovery days +1 (24 h) and +2 (48 h) after training sessions,
165 and compared to rolling baseline data obtained from a minimum of four
166 readings in the prior 7-days ¹⁵. rMSSD was chosen as the HRV variable
167 of interest due to the relationship with vagal activity ¹⁶ and greater
168 reliability compared to other spectral indices ¹⁷.

169

170 **Environmental Monitoring**

171 The training location at which data were collected was a coastal, sub-
172 tropical region (28° S, 153° E) in Australia. The T_a (°C) and RH (%) were
173 measured via a portable weather station (Kestrel 5000, Kestrel
174 Instruments, Pennsylvania, USA), while SR (W/m²) was recorded via
175 pyranometer (MP-100, Apogee Instruments, Utah, USA) at 15-minute
176 intervals during each field-based training session. The devices were
177 mounted on a level tripod 1.5 m above ground in the same location
178 adjacent to the training field. After completion of each training session,
179 data was downloaded to a custom *Microsoft Excel* spreadsheet. To account
180 for the varying duration of training sessions, environmental ‘exposure’
181 was quantified by multiplying session duration by the mean of recorded
182 T_a, RH and SR with data expressed as session means.

183

184 **Statistical Analyses**

185 Training environment data during the experimental period were analysed
186 using one-way analysis of variance (IBM SPSS Statistics, V. 25). Data are
187 presented as mean ± standard deviation with statistical significance set at
188 $p < 0.05$.

189 Relationships between internal and external load and the training
190 environment were analysed using mixed effects linear models via the
191 *Lme4* package in *R Studio* statistical computing software (V. 1.1.442).
192 Mixed-effect linear models were applied to training and recovery variables
193 incorporating the individual as a random effect and heat stress variables as
194 fixed effects using the equation:

195
$$y_i = \beta_1 x_{1i} + \beta_2 x_{2i} + \beta_3 x_{3i} + b_i + \varepsilon_i$$

 196 where y_i is the value of the outcome variable of interest i , β_1 through β_3
 197 are fixed effect coefficients, x_{1i} through x_{3i} are fixed effect variables for
 198 observation i , b_i is the random effects assumed to be multivariate normally
 199 distributed, and ε_i is the error for case i which is also assumed to be
 200 multivariate normally distributed¹⁸. Normality assumptions were
 201 validated using residual and QQ-plots, and the adequacy of the model
 202 structures was determined via residual plots and quantified using standard
 203 measures of intraclass correlations and coefficients of determination. All
 204 variables of interest in the model are reported using standardised
 205 regression coefficients (standardised beta (β)), allowing assessment of
 206 practical significance. Standardised regression coefficients for each
 207 variable were multiplied by the standard deviation of the change in
 208 dependent variable to obtain the absolute change in the units of
 209 measurement¹⁹. Qualitative descriptors for reporting of standardised beta
 210 scores were adopted using a comparable approach to effect size statistics
 211 to interpret the magnitude of the association between heat stress, training
 212 and recovery variables. We interpreted the standardised beta using
 213 threshold values of 0.2 as a small effect, 0.5 as a moderate effect, and 0.8
 214 as a large effect. Multicollinearity between heat stress variables was
 215 established through Spearman's correlation matrix analysis and in the
 216 event that any variables had a multicollinearity $r = > 0.8$ they were
 217 excluded from the model^{20,21}.

219 **Results**

220 **Environmental Conditions**

221 Mean environmental conditions during the experimental period were 30.9
 222 ± 2.1 °C T_a (Range: 26.7 to 34.4 °C), $61 \pm 6\%$ RH (Range: 52 to 75 %) and
 223 718 ± 224 W/m² SR (Range: 239 to 1001 W/m²). Multicollinearity did not
 224 exist between heat stress variables. Wet-bulb globe temperature (WBGT)
 225 during the experimental period was 29 ± 2.5 °C (Range: 24.4 to 32.9 °C).
 226 There was no significant difference in variables of environmental

conditions (Table 1) or training load (Table 2) between training sessions (n=20) throughout the eight-week experimental period.

Insert Table 1 Here.

Insert Table 2 Here.

External Training Load

Increasing SR exposure was associated with a decrease in distance covered (-19.7 m.min⁻¹, $\beta = -0.909$, $p < 0.001$), but there was no significant relationship between changes in RH and m.min⁻¹ at each training session (Figure 1a). In contrast, increasing T_a exposure was associated with an increase in distance covered (19.7 m.min⁻¹, $\beta = 0.911$, $p < 0.001$). There were divergent effects on the change in %HSR completed during training sessions by each of the individual heat stress variables (Figure 1b). Specifically, an increase in SR exposure was associated with a large decrease in %HSR completed during training (-10%, $\beta = -0.953$, $p < 0.001$). There was also a small effect of increasing RH that was associated with lower %HSR (-3.4%, $\beta = -0.319$, $p = 0.010$). However, increasing T_a exposure during training was related to a small increase in percent %HSR (3.5%, $\beta = 0.338$, $P = 0.005$) (Figure 1b).

Internal Training Load

Increasing T_a exposure during training sessions was associated with a concomitant increase in mean HR (4.8 bpm, $\beta = 0.449$, $p < 0.001$). However, there was no relationship between increases in SR or RH exposure and mean HR (Figure 2a). There were no significant relationships between the percent duration of training completed between 65-74% HR_{max} and any heat stress variables during the experimental period ($\beta = -0.04 - 0.12$). Percent duration of training completed between 75-84% HR_{max} was associated with higher RH exposure (4.6%, $\beta = 0.28$, $p = 0.002$), but there was no effect of either SR or T_a exposure on the percent duration of training completed between 75-84% HR_{max}. When RH

258 exposure increased, there was an associated increase in percent duration
259 of training being completed above 85% of HR_{max} (3.9%, $\beta = 0.260$, p
260 <0.001) with a similar result evident with T_a exposure (4.3%, $\beta = 0.287$, p
261 $=0.001$). In contrast, the percent duration of training sessions completed
262 above 85% HR_{max} underwent and associated decrease with increasing SR
263 exposure (-2.9%, $\beta = -0.192$, $p <0.001$; Figure 2b).

264 Increasing RH exposure was associated with an increase in RPE (1.8AU,
265 $\beta = 0.968$, $p <0.001$). In addition, increased T_a exposure was also associated
266 with an increase in RPE, but this was a small effect (0.3AU, $\beta = 0.153$, p
267 $=0.019$). There were no significant relationships between RPE and the
268 level of SR exposure during training sessions.

269 **Recovery**

270 There was no effect of SR exposure on the BM change from pre- to post-
271 session during training, but there was an associated increase in BM loss
272 with increasing RH (-215 g, $\beta = -0.25$, $p <0.001$) and T_a (-160 g, $\beta = -0.35$,
273 $p =0.026$). No effect of any heat stress variable on self-reported Overall
274 Wellness at 24 h ($\beta = -0.198 - 0.003$) and 48 h ($\beta = -0.226 - 0.172$) was
275 evident. However, increasing RH exposure was associated with higher
276 self-reported stress 24 h post-training (-0.11AU, $\beta = -0.24$, $p =0.002$).
277 Increasing RH exposure was not associated with self-reported recovery for
278 the individual variables of Fatigue, Sleep Quality and Mood ($\beta = -0.24 -$
279 0.26). Increasing T_a exposure was associated with a decrease in self-
280 reported Sleep Quality 48 h post-training (-0.57AU, $\beta = -0.58$, $p =0.03$) but
281 no other self-reported recovery variable 48 h post-training was associated
282 with heat stress variables ($\beta = -0.15 - 0.39$). There was no effect of any heat
283 stress variable on rMSSD 24 h post-training ($\beta = -0.152-0.072$; Figure 3a).
284 However, increasing SR exposure was associated with reduced rMSSD 48
285 h post-training (-16.9ms, $\beta = -0.277$, $p =0.019$) but neither RH nor T_a
286 exposure generated any significant effect on rMSSD 48 h post-training ($\beta =$
287 $-0.129-0.288$; Figure 3b).

288

289 **Discussion**

290 This study aimed to determine the individual effects of T_a , RH and SR
291 during physical preparation for professional team-sport. Our data show the
292 importance of considering the impact of the individual heat stress variables
293 contributing to thermal load in isolation. Specifically, we show for the first
294 time that SR is associated with profound effects on ~~the quality of~~ training
295 in team-sport athletes related to reduced self-paced high intensity work
296 performed during the preparation phase of a competitive season. Our
297 findings also show RH is associated with reductions in the level of high-
298 speed running during team-sport training and exerts the largest effect for
299 increasing players rating of perceived exertion. In contrast, increasing T_a
300 was associated with higher work capacity during pre-season training as
301 evidenced by greater external training loads. Accordingly, the novel data
302 from the present study indicates SR and RH each appear to have negative
303 effects on the team-sport athlete, with associated reductions in intermittent
304 high-intensity running capacity and potential to slow recovery between
305 training sessions.

306 Our data are in agreement with the limited number of previous studies
307 investigating the effects of SR on exercise intensity or duration ^{5,22}. Otani
308 and colleagues have previously shown time-to-exhaustion during
309 prolonged endurance exercise is reduced even at moderate SR intensity
310 (500 W/m^2) ⁵ and that self-selected exercise intensity decreases when
311 exposed to increasing levels of SR in hot environments ²². We extend on
312 these findings to show high SR exposure is closely related to impaired
313 high-intensity work capacity during training for a professional team-sport.

314 The associated decrease in high-intensity work performed with increased
315 SR may be related to T_{sk} which is heavily influenced by the external
316 environment ³. SR exposure has been shown to increase T_{sk} in a dose-
317 response manner in thermoneutral ^{23,24} and hot ^{22,25} environments, while
318 having little meaningful effect on core temperature ^{5,24}. The athletes in the
319 current study routinely undertook training under SR intensities equivalent
320 to those reported in previous studies showing increased T_{sk} . Consequently,
321 we suggest that elevated T_{sk} with high SR exposure in the present study
322 was likely a primary factor attenuating the capacity for prolonged high

intensity, intermittent exercise above that associated with high T_a alone. When T_{sk} increases, the core-to-skin temperature gradient narrows and promotes increases in skin blood flow, a decrease in stroke volume, and compromised cardiac output²⁶. During self-paced team-sport training that includes repeated high-speed running in hot conditions, the thermoregulatory response limits exercise intensity to reduce metabolic heat production so that levels of compensable heat strain can be maintained²⁷. While we cannot ascribe cause-and-effect from our data, increased T_{sk} ²⁸ and thermal perceptions of hot skin²⁹ has previously been associated with a decrease in intensity of aerobic exercise performance. Therefore, we propose that the team-sport athletes in the current study downregulated effort and intensity of work in response to higher T_{sk} with increasing SR exposure.

A second major finding of the present study was that RH was also associated with compromised ~~quality of~~ external work and higher internal stress. It is well-established that when RH is low, evaporative cooling is an efficient cooling mechanism and that increasing humidity limits evaporation, as sweat secreted to the skin surface is not easily dissipated to the external environment³⁰. Moreover, in hot conditions, the body will also gain heat from the environment through radiation and conduction¹. In the present study, increasing RH exposure was associated with a small-to-moderate effect on work performed during training but had the largest effect on an individual's perception of exertion. When considering the typical training environment of the present study was hot and humid, the effect of RH on perceived effort and heart rate may have been expected. However, this effect did not appear to decrease external work output to the same extent as SR, indicating there may be incongruence between external and internal load parameters during preparation for team-sport in hot environments with potential implications for training load monitoring of athletes in the heat.

In contrast to the effects of SR and RH, higher T_a was associated with increased work performed and internal load during training sessions. Importantly, an operational construct of the statistical model employed is

that the effects of an increasing individual heat stress variable are determined within a paradigm where the other heat variables in the model remain constant. Under conditions of moderate-to-high T_a (~30-35 °C), it has been purported that the increased muscle temperature with passive heat exposure may promote improved performance capacity for repeated, high-intensity efforts similar to those undertaken in team-sport training³¹. This phenomenon may, at least in part, explain the association between increasing T_a and running performance in the present study. While the associated increase in heart rate with RH likely reflects the effects of heat strain, the association between elevated heart rate and increased T_a may be related to the greater work performed with increasing T_a . Moreover, high levels of aerobic fitness are closely associated with effective heat mitigation, and together with heat acclimation may represent the primary strategies for enhancing exercise capacity in hot conditions³². Participants in our study were heat acclimatised professional team-sport athletes with a high level of aerobic fitness, and a well-developed capability to tolerate stressful training environments. As such, it seems reasonable to suggest that increased muscle temperature could be achieved in trained athletes without or in spite of an excessive rise in core temperature, a response that may also be a prerequisite for improved performance of repeated high-intensity efforts in the heat³³. However, it is possible the magnitude of heat stress from increasing T_a exposure in the present study was insufficient to elicit detrimental effects more commonly associated with heat stress protocols in laboratory settings. Moreover, it is unclear if similar responses to increasing T_a exposure would be observed in different athlete cohorts, geographical locations or whilst undertaking different training protocols.

Intuitively, the physiological strain of undertaking high-intensity team-sport training in hot environments would elicit a significant effect on psychometric wellbeing and parasympathetic activity that is reflected in rMSSD³⁴. Indeed, high-intensity exercise (>50% VO_{2peak}) has been shown to progressively decrease parasympathetic tone³⁵ with potential to increase subjective feelings of fatigue and decrease sleep quality³⁵ during recovery from exercise in hot conditions. The lack of association between

390 heat stress variables and most subjective and objective recovery measures
391 after 24 h recovery was unexpected, although there was a modest
392 association between RH and self-reported stress the day after training.
393 However, a recently published study investigating the recovery time-
394 course in Australian Football has shown that the stress response appears
395 to reach a peak after ~40 h recovery from competition ³⁶. Our data show a
396 small associated decrease in rMSSD 48 h after training with increasing SR
397 exposure, an effect that was not apparent in response to increasing T_a or
398 RH. In addition, T_a was associated with a decrease in self-reported sleep
399 quality 48 h post-training with no other subjective recovery measures
400 associated with heat stress variables. It could be that quantification of
401 acute HRV has limited capacity to detect a meaningful change in the
402 current study and chronic responses analysed over prolonged periods may
403 be more informative ³⁷. Our varied findings may also simply reflect the
404 complexity of interactions in subjective and objective measures of
405 recovery ³⁸, and the most representative sample to detect important
406 relationships is also unknown. Further research is required to determine
407 the relationships between team-sport athlete training load, measures of
408 recovery and heat stress variables, and the effect on athlete well-being
409 during preparation for competition.

410 **Practical Applications**

411 Measuring environmental parameters individually may be more sensitive
412 when assessing the magnitude of heat stress during team-sport training and
413 recovery. Moreover, heat management strategies should not be limited to
414 competition but should include the physical preparation period if
415 maximising the quality and quantity of work completed within team-sport
416 training is desired.

417 **Conclusion**

418 In conclusion, our data is the first to show the effects of individual heat
419 stress variables on team-sport training and the associated acute negative
420 effects on physical performance and recovery parameters with exposure to
421 increasing SR and RH. Indeed, we have demonstrated these effects in heat-

422 acclimatised athletes indicating that heat acclimation alone may be unable
423 to ameliorate reduced acute ~~detrimental~~ responses associated with team-
424 sport training undertaken in hot conditions. Given the reality of a warming
425 climate and the increased prevalence of environmental extremes in many
426 geographical locations, mitigating the effects of heat for team-sport
427 athletes will be increasingly important.

428

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433 Geoffrey Minett, Peter Reaburn, Jonathan Bartlett, and Vernon Coffey
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Table 1. Environmental conditions during the eight-week data collection period. Data are mean \pm standard deviation (n=20 training sessions).

Heat Stress Variable	Weeks 1-8 Average	Weeks 1-8 Median	Weeks 1-8 Range
Ambient Temperature (°C)	30.9 \pm 2.1	31.4	26.7 to 34.4
Relative Humidity (%)	61.7 \pm 6.2	61.2	52 to 75
Solar Radiation (W/m ²)	718 \pm 224	789	239 to 1001
Wind Speed (Kph)	4.0 \pm 1.3	4.1	1.9 to 6.5
WBGT (°C)	29.0 \pm 2.5	29.6	24.4 to 32.9

Table 2. Training session load quantified via GPS, electronic heart monitoring and participant ratings of exertion during the eight-week experimental period for players preparing for Australian Football competition (n= 45). Data are mean \pm standard deviation

Training Variable	Weeks 1-8	Median	Range
Session Duration (min)	70.8 \pm 24.1	60.4	26.7 to 97.7
Session Distance covered (m)	7323 \pm 2853	6417	26.4 to 10197
Average speed (m.min ⁻¹)	102.7 \pm 21.1	102.9	65.3 to 210.9
High-Speed Running (%)	25.0 \pm 10.6	26.7	7.1 to 39.6
Mean HR (bpm)	155.7 \pm 10.7	154.6	140.4 to 163.3
Time >85% HR _{max} (%)	16.9 \pm 15.1	15.5	16.4 to 53.6
RPE (AU)	6.4 \pm 1.9	6.8	2.7 to 9.0
rMSSD +1 (ms)	140 \pm 95	135.7	60.3 to 183.4
rMSSD +2 (ms)	140 \pm 95	136.6	60.3 to 183.4

HR, heart rate; RPE, rating of perceived exertion; rMSSD, root mean square of the successive differences.

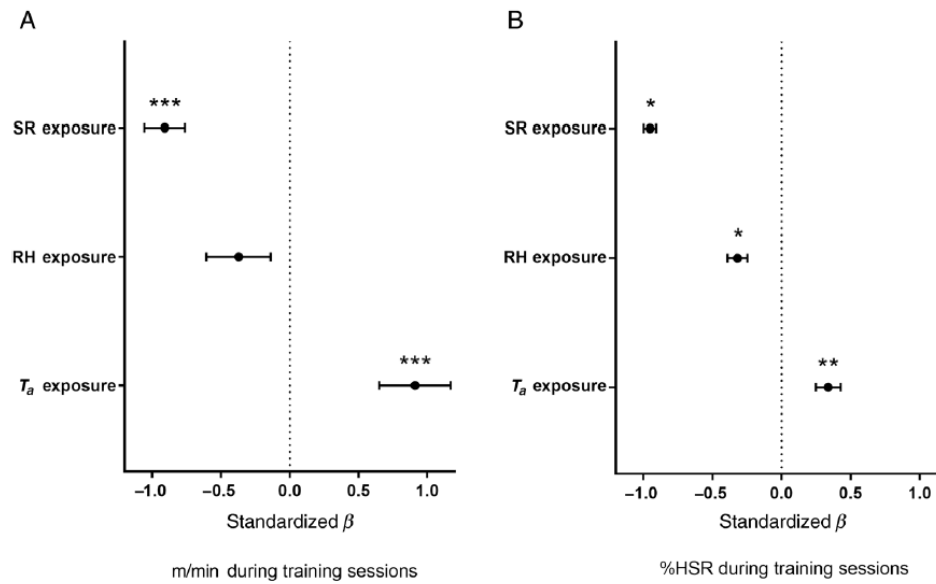


Figure 1 — Standardized coefficient relationships between (A) distance covered (in m/min) and heat-stress variables and (B) %HSR and heat-stress variables during an 8-week experimental period for players preparing for Australian Football competition (N = 45; 20 training sessions). SR indicates solar radiation; RH, relative humidity; T_a , ambient temperature; %HSR, percentage high-speed running. * $P < .05$. ** $P < .01$. *** $P < .001$.

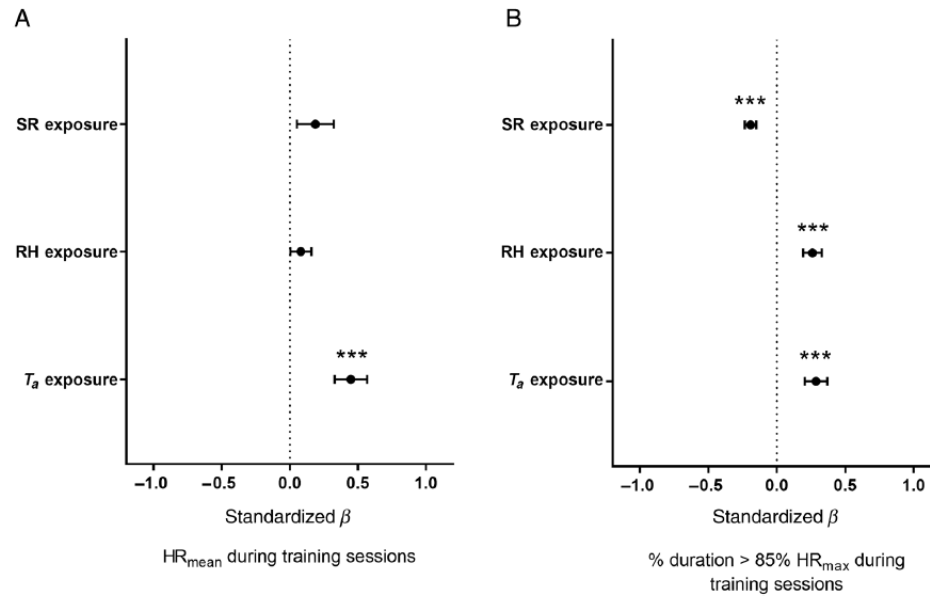


Figure 2 — Standardized coefficient relationship between (A) HR_{mean} and heat-stress variables and (B) percentage duration above 85% of maximal heart rate and heat-stress variables during an 8-week experimental period for players preparing for Australian Football competition (N = 45; 20 training sessions). HR_{mean} indicates mean heart rate; SR, solar radiation; RH, relative humidity; T_a , ambient temperature. *** $p < .001$.

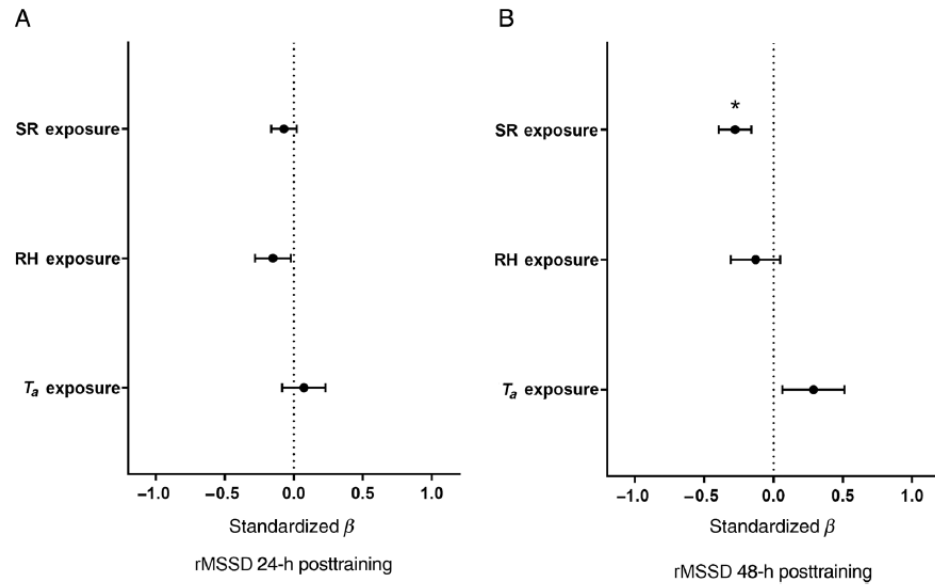


Figure 3 — Standardized coefficient relationship between heart-rate variability (rMSSD) and heat-stress variables following (A) 24- and (B) 48-hour recovery from training sessions during an 8-week experimental period for players preparing for Australian Football competition (N = 45; 20 training sessions). SR indicates solar radiation; RH, relative humidity; T_a , ambient temperature; rMSSD, root mean sum of the squared differences. *P < .05.